

## FITC Anti-Human TNF- $\alpha$ Antibody

<b>Catalog Number:</b>	113505, 113506
<b>Size:</b>	25 tests, 100 tests
<b>Target Name:</b>	TNF- $\alpha$ , TNF-alpha, Tumor necrosis factor- $\alpha$ , Macrophage cytotoxic factor (MCF)
<b>Regulatory Status:</b>	RUO

### PRODUCT DETAILS

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<b>Clone:</b>	Infliximab
<b>Application:</b>	Flow Cytometry
<b>Reactivity:</b>	Human
<b>Format:</b>	FITC
<b>Isotype:</b>	Human IgG1
<b>Antibody Type:</b>	Monoclonal
<b>Formulation:</b>	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA
<b>Protein Concentration:</b>	Supplied at a lot-specific concentration.
<b>Storage&amp;Handling:</b>	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.
<b>Recommended Usage:</b>	For flow cytometric staining, it is recommended to use 5 $\mu$ L of this reagent per 0.5-1.0 million cells in a 100 $\mu$ L volume. Optimal reagent performance should be determined by titration for each specific application. FITC has an excitation max at 493 nm and an emission max at 525 nm.
<b>Excitation Laser:</b>	Blue Laser (488 nm)
<b>Isotype Control:</b>	301205

### BACKGROUND INFORMATION

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TNF $\alpha$  (tumor necrosis factor alpha) is a potent pro-inflammatory cytokine that plays a central role in immune regulation, host defense, and inflammation. It is primarily produced by activated macrophages, T cells, and other immune cells in response to infection, injury, or immune stimulation. TNF $\alpha$  mediates a wide range of biological effects, including induction of fever, activation of endothelial cells, promotion of leukocyte recruitment, and regulation of cell survival, apoptosis, and necrosis.

Structurally, TNF $\alpha$  is initially synthesized as a type II transmembrane protein (membrane-bound TNF $\alpha$ ) that forms stable homotrimers. It can be cleaved by the metalloprotease TACE (ADAM17) to release a soluble trimeric form. Both membrane-bound and soluble TNF $\alpha$  are biologically active, although they may have distinct functional roles. The trimeric structure is essential for binding and activating its receptors.

The primary receptors for TNF $\alpha$  are TNFR1 (p55, CD120a) and TNFR2 (p75, CD120b). TNFR1 is widely expressed and contains a death domain that can trigger apoptosis or activate NF- $\kappa$ B signaling pathways, leading to inflammation and cell survival. TNFR2 is more restricted in expression and is mainly involved in immune regulation and tissue repair. The interaction between TNF $\alpha$  and its

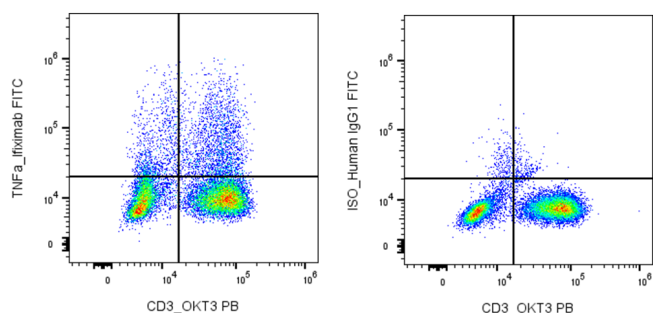
receptors initiates complex signaling cascades that determine cellular outcomes.

In disease, TNF $\alpha$  is a key driver of chronic inflammatory and autoimmune conditions, including rheumatoid arthritis, inflammatory bowel disease, psoriasis, and ankylosing spondylitis. Excessive or dysregulated TNF $\alpha$  production contributes to tissue damage and disease progression. TNF $\alpha$  is also involved in cancer, infection, and sepsis, where it can have both protective and pathological effects.

Therapeutically, TNF $\alpha$  is one of the most successfully targeted cytokines in modern medicine. Anti-TNF biologics, such as monoclonal antibodies and receptor fusion proteins, have revolutionized the treatment of inflammatory diseases by neutralizing TNF $\alpha$  activity. These therapies reduce inflammation and improve clinical outcomes, although they may increase susceptibility to infections due to immune suppression.

## PRODUCT DATA

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Human peripheral lymphocytes stimulated with PMA/Ionomycin were stained with PB Anti-Human CD3 clone OKT3, fixed, permeabilized and then stained with FITC Anti-Human TNF $\alpha$  biosimilar Infiximab (left) or an isotype control (right).

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